by 31% (5). Because stroke is a major cause of dementia, statins must be considered potentially valuable agents for preventing cognitive decline in patients with risk factors for atherosclerosis or dementia.

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Does Inhaled Nitric Oxide Support the Hemodynamic of Spontaneous Breathing Patients With Cardiogenic Shock Related to Right Ventricular Myocardial Infarction?

We read with great interest the echocardiographic study published by Inglessis et al. (1) concerning hemodynamic effects of inhaled nitric oxide (NO) in right ventricular myocardial infarction (RVMI) and cardiogenic shock (CS). They found that inhaled NO results in acute hemodynamic improvement when administered to patients with RVMI and CS.

We have a major concern with these results. Indeed, although 10 of 13 patients were under positive pressure ventilation, the investigators leave the reader with the feeling that inhaled NO results in significant hemodynamic improvement and a reduction of right to left shunting when administered to all types of patients.

In our opinion, we may expect that the observed NO effect could not be shown in spontaneous breathing patients. Indeed, as stated by the researchers, breathing NO is thought to increase pulmonary venous return and left ventricular filling pressure when cardiac output is decreased (2). Because positive pressure ventilation acts as a circulatory pump (3) and decreases left ventricular transmural pressure, acute left ventricular failure may occur when the lungs are not mechanically assisted.

In this setting, we suggest to Inglessis et al. (1) not to extend their conclusions regarding the hemodynamic inhaled NO effects to spontaneous ventilated patients with acute RVMI and CS.

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REPLY

We thank Dr. Bendjelid for his interest in our work (1). We agree that the majority of our patients were studied while undergoing positive pressure ventilation. As only three patients in our study population did not require mechanical ventilation, our ability to extrapolate our results to patients with right ventricular myocardial infarction (RVMI) not receiving mechanical ventilation is limited. Nonetheless, there was no difference in the improvement in cardiac index observed between those patients breathing nitric oxide (NO) who were mechanically ventilated and those who were not.

Dr. Bendjelid also raises the concern that positive pressure ventilation may act to prevent the development of acute left ventricular (LV) failure that may occur during NO inhalation, and that LV failure may arise in nonventilated patients. Left ventricular filling pressures have been found to increase during NO inhalation in patients with severe LV systolic dysfunction (2,3). The RVMI patients in our study had primarily RV dysfunction, and the degree of LV dysfunction was not as severe as in those patients in whom the pulmonary capillary wedge pressure (PCWP) has been reported to increase during NO inhalation. Furthermore, we excluded patients with a PCWP >25 mm Hg from study. In the three nonventilated RVMI patients in our study, we did not observe an increase in their PCWP while they were breathing NO for 10 min.

In future studies of the effects of sustained NO inhalation in RVMI patients, it will be important to observe the hemodynamic effects of this agent in patients who receive positive pressure ventilation as well as those who do not. Patients with severe LV systolic function should be monitored carefully during chronic NO inhalation because of the possibility of their developing pulmonary venous hypertension.